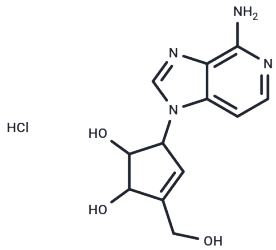
Data Sheet (Cat.No.T6360)

TargetMol

3-Deazaneplanocin A HCl

Chemical Properties



CAS No. : 120964-45-6

Formula: C12H15ClN4O3

Molecular Weight: 298.73

Storage: Store at low temperature
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	3-Deazaneplanocin A HCl is a synthetically derived inhibitor of the histone methyltransferase (EZH2) and S-adenosylhomocysteine hydrolase (SAHH). By regulating epigenetic methylation pathways, 3-deazaneplanocin A induces apoptosis and inhibits tumor cell proliferation, demonstrating significant antitumor activity in various tumor models. 3-deazaneplanocin A HCl can be used in research on epigenetic regulation, tumorigenesis and progression, and stem cell differentiation.
Targets(IC50)	Histone Methyltransferase,Virus Protease
In vitro	**Methods**: 3-Deazaneplanocin A HCl (0.25-2.0 μM) was added to OCI-AML3 and HL-60 cells and treated for 24, 48, or 72 hours. Annexin V/PI double staining flow cytometry and Western blot analysis for PARP cleavage were performed. **Results**: 3-Deazaneplanocin A HCl induced apoptosis in a dose-dependent manner, with OCI-AML3 being more sensitive than HL-60. [1] **Methods**: Tumor spheroids formed by PANC-1, MIA-PaCa-2, and LPC006 cells (cultured in serum-free stem cell medium for 10-14 days) were treated with 3-Deazaneplanocin A HCl (5 μM) for 72 hours. Tumor spheroid volume was measured microscopically; CD133 mRNA was detected by RT-qPCR. **Results**: 3-Deazaneplanocin A HCl significantly reduced tumor spheroid volume. [2] **Methods**: Rat primary HSCs; JS1 cells; LX-2 cells were treated with 3-Deazaneplanocin A HCl (1 μM) for 48-96 hours. Western Blot analyzed EZH2, H3K27me2/3, α-SMA, COL1A; CCK-8 assayed cell viability; flow cytometry assessed cell cycle and apoptosis; SA-β-Gal assay for senescence; TUNEL assay for apoptosis. **Results**: 3-Deazaneplanocin A HCl treatment maintained HSCs in a more quiescent state, reduced EZH2, H3K27me2/3, α-SMA, and COL1A expression levels, leading to cell cycle arrest at S and G2 phases, decreased viability, increased senescence, and enhanced early apoptosis.[3]
In vivo	**Methods**: NOD/SCID mice were injected with 5×10^6 HL-60 cells via the tail vein to establish an AML model. Drug administration commenced on day 7 post-cell injection (control group: solvent; 3-Deazaneplanocin A HCl monotherapy group: 1 mg/kg, intraperitoneal injection, twice weekly for 2 weeks). Mouse survival times were recorded and Kaplan-Meier survival curves were plotted. **Results**: The median survival time in the 3-Deazaneplanocin A HCl-treated group (43 days) was significantly longer than that in the control group (36 days). [1]

In vivo	**Methods**: To investigate the therapeutic effect of 3-Deazaneplanocin A HCl in liver fibrosis, BALB/c mice were used. Hepatic fibrosis was induced by intraperitoneal injection of CCl_4 (for 4 weeks), concurrently with intraperitoneal injection of 3-Deazaneplanocin A HCl (1 mg/kg) twice weekly; The control group received DMSO injections for 4 weeks. **Results**: 3-Deazaneplanocin A HCl significantly reduced hepatic levels of EZH2, H3K27me3, α-SMA, Col1a1, and Mmp2, markedly decreased collagen deposition and α-SMA-positive areas, and significantly lowered serum ALT and AST levels. [3] **Methods**: Rats implanted with microdialysis probes in the nucleus accumbens (monoamine/adenosine) received intraperitoneal injections of 3-Deazaneplanocin A HCl (0.1, 1.0, 10 mg/kg) during the light phase. Dialysate was collected for 4 hours post-administration, and dopamine (DA), norepinephrine (NE), epinephrine (EP), serotonin (5-HT), adenosine (AD), and acetylcholine (ACh) were quantified by high-performance liquid chromatography (HPLC). **Results**: 3-Deazaneplanocin A HCl significantly increased extracellular levels of all neurochemicals in a dose-dependent manner. [4] **Methods**: Female SD rats were established with bone cancer pain by intraosseous injection of Walker 256 breast cancer cells (approximately 1×10^5 cells/10 μL) into the tibial bone marrow cavity. 3-Deazaneplanocin A HCl (5 nM, 20 nM, 40 nM) was administered via intrathecal injection on days 3–5 post-injection, with observation of pain threshold (PWT) and spontaneous activity. **Results**: 5 nM was ineffective; 40 nM increased spontaneous activity (potentially a side effect); 20 nM was both effective and safe.[5]

Solubility Information

Solubility	H2O: 50 mg/mL (167.38 mM),Sonication is recommended. DMSO: 150 mg/mL (502.13 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.3475 mL	16.7375 mL	33.475 mL
5 mM	0.6695 mL	3.3475 mL	6.695 mL
10 mM	0.3348 mL	1.6738 mL	3.3475 mL
50 mM	0.067 mL	0.3348 mL	0.6695 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Fiskus W, et al. Combined epigenetic therapy with the histone methyltransferase EZH2 inhibitor 3-deazaneplanocin A and the histone deacetylase inhibitor panobinostat against human AML cells. Blood. 2009 Sep 24;114(13):2733-43.
Avan A, et al. Molecular mechanisms involved in the synergistic interaction of the EZH2 inhibitor 3-deazaneplanocin A with gemcitabine in pancreatic cancer cells. Mol Cancer Ther. 2012 Aug;11(8):1735-46.
Jiang Y, et al. Histone H3K27 methyltransferase EZH2 and demethylase JMJD3 regulate hepatic stellate cells activation and liver fibrosis. Theranostics. 2021 Jan 1;11(1):361-378.
Murillo-Rodríguez E, et al. Sleep and Neurochemical Modulation by DZNep and GSK-J1: Potential Link With Histone Methylation Status. Front Neurosci. 2019 Mar 15;13:237.
Chen H, et al. Evidence of the Involvement of Spinal EZH2 in the Development of Bone Cancer Pain in Rats. J Pain Res. 2021 Nov 23;14:3593-3600.